

Arterial Complications of the Thoracic Outlet Syndrome

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Objectives: Arterial complications due to compression of the thoracic outlet are uncommon. The objective of this study was to review our fairly extensive experience with this problem with particular reference to its management.

Methods: Patients entered into the Vascular Clinic database were reviewed over an 11 year period. Twenty six records were found. In 24 patients the vasculopathy was caused by a cervical rib (complete in 15) and in two by an anomaly of the first rib. In all patients the basic arteriopathy was a fibrous structure with a post-stenotic aneurysm in 13. Seventeen presented with a fixed pulse deficit; 13 had a palpable aneurysm and 12 had distal embolisation.

Results: Two patients refused operation. In 22 with cervical rib, the rib was removed via a supraclavicular incision, an anterior scalenectomy was performed and the arterial pathology repaired on its merit, usually by vein graft replacement or bypass. In two with first rib anomalies these were resected by the transaxillary route. Twenty three patients have been followed for between 3 months and 10 years; 20 are cured and three have residual claudication.

Conclusions: Our results show that simple excision of the cervical rib via the supraclavicular route together with vascular reconstruction is adequate. This is in disagreement with the view of those who advocate routine excision of the first rib in addition to cervical rib excision.

Key Words: Thoracic outlet syndrome; Cervical rib; Arterial complications; Ischaemic hand

Introduction

The incidence of thoracic outlet syndrome is difficult to determine. Patients usually present with compression of the lower trunk of the brachial plexus leading to neurological symptoms in the distribution of the C8 and T1 dermatomes. Complications of subclavian artery compression have been reported but few have wide experience. The serious nature and consequences of vascular complications in patients who have recently presented to our vascular service with thoracic outlet compression has prompted the present report.

Patients and Methods

The records of all patients referred to the Vascular Service of the University of Natal Hospitals with arterial complications of thoracic outlet syndrome during the last 11 years were culled from a computerised clinical data base. Twenty six records were

found and analysed. Six were male and the ages ranged from 20 to 50 years and averaged 29 years. In 24 cases the vasculopathy was caused by compression of the subclavian artery between a cervical rib and the insertion of the scalenus anterior muscle. In 15 patients the rib was complete and articulated on the first rib (Fig. 1); nine were bilateral. In seven there was a fibrous band extending from the end of a partial cervical rib to the scalene tubercle and in two patients who declined operation this was presumed to be the pathology as both had radiological evidence of a partial cervical rib. Two patients had an anomaly of the first rib, which articulated on the second rib resulting in compression of the artery.

The arterial pathology was a post-stenotic aneurysm in 13 patients and a fibrous stricture in the remaining 13. Four patients had superimposed acute distal thrombosis. The clinical presentation of these patients is summarised in Table 1. Seventeen patients presented with a fixed pulse deficit.

A bruit in the supraclavicular region was the next most frequent finding and in 13 patients an aneurysm was palpable. Complications related to thrombus formation were encountered on twelve occasions, seven patients had digital vessel embolism and four had acute occlusion of the subclavian and axillary

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arteries and presented with critical upper limb ischaemia.

Nine patients presented with forearm claudication, eight had symptoms attributable to brachial plexus compression with parasthesia and diminished sensation in the C8/T1 dermatome distribution. Only two patients gave a history suggestive of Reynauds phenomenon. All patients had thoracic inlet X-rays and angiography to confirm the vasculopathy.

Operative Management and Results

The operative management is summarised in Table 2. Two patients with claudication declined surgical therapy. Both had a fixed pulse deficit with stenosis of the subclavian artery on angiography; 24 were therefore submitted to operation.

Through a standard supraclavicular approach, all had an anterior scalenectomy. Twenty two had excision of the cervical rib. In 15 patients the rib was complete and was removed at the articulation with the first rib and divided as close to its proximal articulation on the seventh cervical vertebra as possible. In most patients it was possible to remove the entire rib in this fashion. In six patients the cervical rib was found to be in varying stages of development with a fibrous band attaching it to the first rib. The arterial pathology constituted a trauma induced fibrous stricture at the compression site in every case. In 13 patients this was associated with a post-stenotic dilatation (aneurysm). In two patients an abnormal

first rib which articulated anteriorly on the second rib was excised through a trans axillary approach, after the scalenectomy and mobilisation of the subclavian artery had been completed through a supraclavicular incision.

Arterial reconstruction entailed excision of the lesion with an interposition graft, in the majority using reversed saphenous vein graft. In three patients with acute upper limb ischaemia resulting from extensive thrombosis of the subclavian and axillary vessels a long reversed saphenous vein graft was placed between the subclavian and the proximal brachial artery. One patient was deemed to have irreversible ischaemia of the arm at the time of presentation, by virtue of rigid muscle compartments and fixed skin staining and a primary above elbow amputation was performed. Nine patients had a cervical sympathectomy performed as an ancillary procedure in an attempt to improve digital vessel perfusion.

There was no operative mortality. Five patients complained of significant paresthesias in C8/T1 distribution most likely due to brachial plexus traction injury during the operation. Twenty three patients who have had arterial reconstructions performed have been followed for between 3 months and 10 years. Twenty remain asymptomatic while three have residual claudication. All of the latter follow long subclavian to brachial grafts, all of which have occluded. In one patient this symptom is incapacitating and she remains a major management problem. In two patients the contralateral cervical rib has been excised, as they have subsequently developed neurological symptoms.

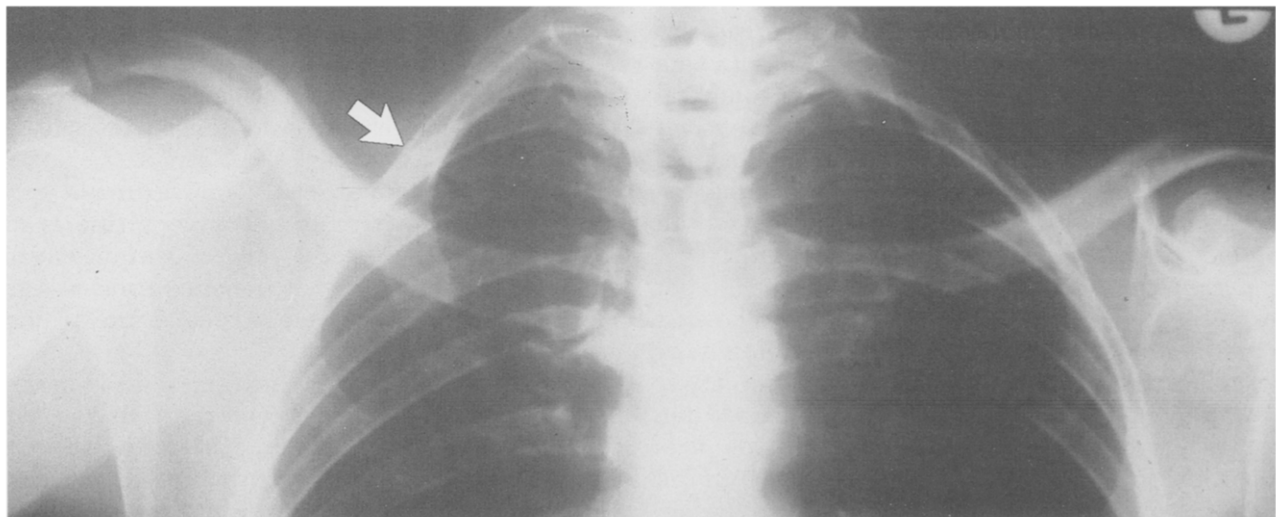


Fig. 1. Radiograph of the chest showing a complete cervical rib articulating on the first rib. (Arrowed).

Table 1. Clinical presentation of patients with cervical rib and arterial complications (n=26)

Pulse deficit	15
Bruit	15
Pulsatile mass	13
Claudication	9
Digital vessel embolisation	7
C ₈ T ₁ Sensory disturbance	8
Acute upper limb ischaemia	4
Reynauds phenomenon	2

Table 2. Operative management of patients with cervical rib and arterial complications (n=24)

Excision cervical rib	22
Arterial reconstruction	23
Excision and interposition graft (16)	
Patch angioplasty (4)	
Subclavian-brachial bypass (3)	
Cervical sympathectomy	9
Digital amputation/debridement	3
Primary above elbow amputation	1
First rib resection	2

Discussion

Vascular complications in patients presenting with the thoracic outlet compression syndrome are uncommon and have been estimated to involve the subclavian vein in 1.5% of patients and the subclavian artery in 0.5%.¹⁻⁴ Recent experience with several patients who have presented to our Vascular Surgical Service with serious complications related to subclavian artery lesions secondary to thoracic outlet compression has prompted the present review which amounted to 26 patients seen over the last decade. This represents one of the largest series presenting with this problem published to date. Raymond *et al.*⁵ reviewed the anatomical anomalies encountered in their extensive experience of 200 patients submitted to surgery for symptoms of thoracic outlet compression. Although they did not specifically address vascular complications it is of interest to note that only 8.5% of their patients had a cervical rib or an anomaly of the first rib. First rib anomalies included synostosis of the first and second ribs or a bifid first rib. Not all patients with neurological symptoms due to thoracic outlet syndrome are referred to our Vascular Service and it is not possible to provide any meaningful data on what proportion of these patients present with arterial complications in our practice. The present study specifically addresses arterial complications and the most frequently encountered lesion causing compression of the subclavian artery was a complete cervical rib articulating on the scalene tubercle of the first rib, which occurred in 60% of our patients. This is in

agreement with the findings of others.^{6,7} The next most frequent pathology was a partially developed cervical rib with a fibrous band extending from its tip to the scalene tubercle on the first rib, while two of our patients had an osseous anomaly of the first rib. Although arterial compression due to hypertrophy of the scalenus anterior muscle has been reported we have not encountered this in our own practice.

Sher *et al.*,¹ based on the original description of Lewis and Pickering⁸ described various stages in the development of arterial lesions due to arterial compression. Stage I comprised compressive stenosis with post stenotic dilatation; Stage II aneurysm formation with intimal damage and mural thrombosis and Stage III the complications of embolisation and thrombosis. In our patients in every case the arterial pathology was related to a fibrous stricture at the junction of the second and third parts of the subclavian artery which was associated with a post stenotic dilatation in half the patients. This had resulted in embolic complications from a thrombus containing aneurysm in nearly a third of the patients. (Stages II and III)

It would appear that the diagnosis of arterial complications of thoracic outlet compression is only made once there is structural damage to the artery. Almost half of the patients in the present series presented with severe ischaemic symptoms necessitating amputation or digital debridement. While the emphasis should be placed on early diagnosis this may prove difficult as a fair proportion of a totally asymptomatic population exhibit disappearance of rest pulses on provocative testing such as shoulder bracing (Adson's Test) or abduction and external rotation of the shoulder.^{1,7,8} By the same token it would appear that the incidence of significant vascular damage due to thoracic outlet syndrome is low. A compromise would be to suggest that any patient who presents with symptoms suggestive of circulatory insufficiency in the upper limb such as claudication, Reynauds' type symptoms, or evidence of digital vessel embolisation should be thoroughly investigated for evidence of thoracic outlet compression. This would include thoracic inlet X-rays to reveal gross bony abnormalities, segmental doppler pressures and angiography. More recently we have used the Duplex doppler to assess subclavian artery lesions with some success and this has developed into a useful diagnostic modality.

With regard to operative management the general principle embraces decompression of the thoracic outlet coupled with repair of the arterial lesion on its merits. Brown and Charlesworth⁹ suggest that cervical rib excision alone is inadequate therapy for arterial problems. It is difficult to accept this conclusion on the

basis of their experience with only four patients in this category. Davies *et al.*¹⁰ report on 14 patients in their series with arterial symptoms due to cervical rib, and all improved following simple excision of the rib. Support for the contralateral view is found in the report by Thompson and Webster¹¹ who had 11 patients with a cervical rib causing arterial problems. In their experience first rib resection constituted standard therapy which they conclude is optimal although the evidence for this is far from conclusive as none were treated by simple cervical rib excision. In our experience the optimum approach is via a supraclavicular incision with anterior scalenectomy. Should the obstructing lesion prove to be a cervical rib we believe that scalenectomy coupled with complete excision of the rib, including its periosteum, provides adequate decompression with good long term results. It has, however, been pointed out by Edwards *et al.*¹² that a possible cause of failure of this method of treatment of cervical rib is a residual bony promontory on the first thoracic rib related to the articulation.

In our two patients with an anomaly of the first rib this was excised via a trans-axillary approach while the arterial pathology was repaired via the standard supraclavicular route. Based on the results in this series we do not advocate routine excision of the first rib coupled with cervical rib excision as advocated by others.^{1,9,11,13} Similarly transection or excision of the clavicle under these circumstances appears to have little place, if any.^{8,10,14,16,17} The arterial lesion should be treated on its merits with excision and interposition autogenous vein grafts. However, occasionally a patch angioplasty may suffice. In our hands long bypasses from the subclavian to the brachial or even more distally has occluded within weeks due to distal runoff problems. This had in every case resulted from repeated episodes of embolisation into the smaller distal vessels.

In general the results of treatment with the exceptions mentioned have been good in that they have been cured of their symptoms and this has proved durable during the period of follow up (3 months to 10 years). A dilemma that persists is the management of the asymptomatic contralateral complete cervical rib, found in nine of our patients. Our approach has been to follow them up with a view to more aggressive

management should any symptoms arise. Excision has become necessary in only two patients.

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